PHILADELPHIA NEUROLOGICAL SOCIETY.

January 28, 1902.

The Vice-president, Dr. Charles S. Potts, in the chair.

A Case of Paralysis of the Face, Upper and Lower Limbs on the Right Side; and of the Tongue and Muscles of Mastication on the Left Side.—This case was presented by Dr. Wm. G. Spiller. Paralysis of one of the ocular muscles was also present, but Dr. W. C. Posey had had difficulty in determining which muscle was paralyzed. The case was diagnosticated as one of multiple syphilitic lesions.

Dr. Edward A. Shumway said that he had made a brief examination of the patient and found that she had diplopia which increased towards the left, and increased markedly when the patient looked up and to the left. This indicated interference with the left superior rectus or the right inferior oblique. The patient's answers were uncertain, but there was apparently a paresis of the right inferior oblique.

A Case of Superior Tabes with Symptoms of Paretic Dementia.—This case was presented by Dr. Max H. Bochroch.

Dr. J. K. Mitchell said that tabes limited, or almost limited to the arms was very little mentioned in the text-books, Erb, Leyden, Vulpian and Hammond barely referring to it or omitting it altogether.

Dr. Weir Mitchell in 1888 reported the first case observed in the United States, the speaker the second one in 1894. It was curious that Dr. Bochroch's case and both these others should be in men making much use of their arms in their trades, as carpenters and masons.

In the speaker's case the trouble began in the right forearm and for a year or more was limited to that part. After six years the ataxia and impairment of sensation which were very great were still strictly limited to the forearms. There were some subjective sensations of discomfort in the feet. The gait was good, but the knee-jerk was totally absent. He walked without difficulty and had no lancinating pains except in the arms. When he was last heard of, three years later, the disease had progressed no further.

Dr. J. Madison Taylor referred to a case of Dr. Weir Mitchell which he had seen, one which had passed through the hands of a celebrated neurologist in London without the disease being recognized. The patient consulted Dr. S. Weir Mitchell for another matter. The tabes in the lower limbs was very slight. The ataxia in the arms was well marked. There were lancinating pains in the arms. The man had used his arms largely in out-door sports. He had first noticed diminution in his ability to use firearms. The general health was thoroughly good. Improvement set in in the arms and the legs grew worse. This was not the case reported by Dr. John K. Mitchell, but a second one occurring in his father's practice.

Dr. William G. Spiller said that Leyden had spoken of cervical tabes in 1876. Dr. Spiller referred to a case, the specimens from which had been sent to him about one year ago by Dr. S. S. Cohen. Symptoms suggesting syringomyelia had been present. It was a case of superior and bulbar tabes, and was reported at the meeting of the American Neurological Association last Spring.[1] A number of clinical cases of superior tabes have been reported, but very few (probably not more than eight or nine), with necropsy. The case which he had studied was distinctly one of superior tabes associated with bulbar tabes. The lower cervical and the upper thoracic roots were degenerated. Strictly speaking, the term cervical tabes is a misnomer. The disease is not confined to the cervical cord, but in all the cases with necropsy studied the upper part of the thoracic cord has been involved. In the case reported by Drs. Cohen and Spiller, the lumbar cord was not diseased. The patient had been a carpenter, and therefore had used his upper limbs more than his lower.

Dr. F. S. Pearce suggested, in regard to etiology, the possibility that the primary cause in some cases of superior tabes was a latent meningitis. Many cases with symptoms of amyotrophic lateral sclerosis are probably of this character.

Dr. Alfred Gordon referred to the relations between tabes and paresis. A study of the literature shows that the association of paresis and tabes is more common than is generally supposed. The principal objection of those who think that tabes and paresis are not related, is the fact that tabes to arrive at its full development requires ten to twenty years, while paresis requires only a few years. A reference to the literature shows that this is not so. Barthelemy in 1878 reported a number of cases. Fournier has also collected five hundred cases of tabes, and has followed up a great many of them. In a large proportion of these cases, paresis developed after the onset of tabes.

Ocular symptoms are found in both diseases. The motor incoördination of tabes is analogous with incoördination of mentality in paresis. The etiology is also analogous. Paresis and tabes are often found in different members of the same family. The progressive character and the impossibility of arrest are analogous in the two diseases. An important fact is that those who have followed up their cases have always witnessed symptoms belonging to paresis. In the cases of incipient tabes reported by Westphal, Strümpell and Flechsig, symptoms of paresis developed very rapidly.

With regard to the knee-jerk in superior tabes, Westphal contends that if in superior tabes the knee-jerk disappears, it shows that the lumbar cord is involved. That, however, is not always the case. In one case in which the knee-jerks were absent, the autopsy showed no involvement of the lumbar cord. Symptoms of paresis were also present in this case.

Dr. Gordon referred to a case reported by Raymond in which a diagnosis of tabes had been made. Shortly before death cerebral symtoms developed, and the autopsy showed that it was a case of paresis.

Dr. Charles K. Mills said that he had come to the conclusion that the views of Raymond are correct that tabes and general paresis in their pure forms are essentially the same, that is to say, pathologically and patogenetically. The subject is one which requires somewhat elaborate discussion. He believed that the trend of opinion is in the direction referred to. He had, however, seen cases in which after many years of tabes, the patients became demented, but the dementia was not of the tabetic paretic type. In almost all cases of tabes of long duration, some cerebral symptoms and lesions can be found; and it is rare to find general paresis in which some spinal symptoms and lesions do not exist. It is the localization and the diffusion of the lesions which give us the clinical types—spinal in the one case, cerebral in another, and cerebro-spinal in a third.

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[1] American Journal of the Medical Sciences, August, 1901.

Dr. William Pickett said the question of paresis in Dr. Bochroch's case was of interest. While cases of paresis supervening upon tabes dorsalis are sometimes spoken of as *ascending* paresis, this is not strictly correct, as the disease does not progress from tabes dorsalis to "tabes of the brain" by a continuity of lesion. If it did, we might expect these cases of cervical tabes to develop into paresis more frequently. Out of 149 cases of paresis which he had recently studied there were 5 of the ascending type. These five were all long-standing cases of tabes of the lumbo-thoracic region, and in 4 of them it was the classic form of paresis which developed, the fifth being a simple demented case. He had also seen dementia develop in long-standing cases of tabes, but this was the simple secondary dementia which we see in various forms of cord disease.

He also referred to an instance of paranoia in a typical case of tabes of some fifteen years' duration, with Charcot's joints. The man had hallucinations of hearing, persecutory delusions and considerable exaltation.

In Dr. Bochroch's case the prognosis would be very different according to whether the mental signs are referred to incipient paresis or to simple secondary dementia.

Dr. Max H. Bochroch remarked that in his case there were some of the classical symptoms of paresis, such as fibrillary contractions of the tongue and muscles of the face, hesitation in speech and slow mentality. These symptoms coming on during a short period of time, eighteen months, inclined him to regard the case as one of paresis, with beginning tabes.

A Case of Progressive Spinal Muscular Atrophy in which the Atrophy Began in the Extensors of the Hand and Fingers.—This case was shown by Dr. C. S. Potts.

Dr. Charles K. Mills said that such cases as this, although of a common type, were well worth presenting, as they caused us to be guarded with regard to positive opinions about the etiology of cases somewhat similar. This man had some features of lead paralysis. He retained the use of the supinator on one side, while the muscles supplied by the posterior interosseous were largely wasted and weakened. At one time, he had very little atrophy of the muscles of the hand. At some period in its evolution a case of chronic degenerative disease may present a symptom-picture closely resembling that found in toxic disease.

A Case of Facial Tic in which each Series of Contraction was followed by Complete Paralysis in the Facial and Hypoglossal Distribution on the Same Side.—This case was reported by Dr. A. P. Francine and Dr. D. J. McCarthy. Dr. D. J. McCarthy said that the primary attack was probably hysterical, but following that the clonic convulsions of the face developed. He had seen the patient in an attack which came on with clonic movements of the right side of the face lasting from half a minute to one minute, and was followed by complete paralysis of the right side of the face and tongue, continuing from two to five minutes. This paralysis disappeared gradually, leaving some weakness. The ocular symptoms were negative, the reflexes were normal. Some hyperesthesia was present on the right side. He regarded the case as organic, with a cortical lesion, or as one with involvement of the seventh and twelfth nerves; more probably however as one of cortical lesion.

Dr. Charles K. Mills remarked that the history would indicate that the case was one of small cortical lesion in the facial area, as shown by the recurrence of the facial spasm, and the clonic type of the spasm, followed by paralysis (Hughlings Jackson exhaustion paralysis). This form of paralysis is usually due to lesion which does not entirely destroy the cortex, but by irritation causes a severe discharge from certain cortical areas. The hypesthesia might be due to a similar discharge through the sensory cortical neurones which send their processes to the motor cortex, or it might be a hysterical epiphenomenon. He did not lay any stress on the absence of optic neuritis in cases of small tumors of the cortex.

Dr. F. S. Pearce referred to a case which had been under his care, in which there were epileptic seizures involving the left side, particularly the arm, and these were followed by paralysis. The man probably had tuberculosis of the lung although no bacilli were found. There was no optic neuritis. Operation over the right motor cortex was advised, but the patient went home and died. No autopsy was made, but the speaker thought that there probably was a tuberculous abscess.

Dr. William G. Spiller thought that the diagnosis of a small cortical lesion was probably correct. It was noteworthy that there was no disturbance of speech. If in this case there were a basal lesion, it was strange that only the seventh and twelfth nerves were affected. While this was possible in meningitis, he considered it more likely that the lesion was cortical.

A Case of Poliomyelitis with Increased Knee-jerk. This case was shown by Dr. F. Savary Pearce. Dr. Charles K. Mills considered this to be a case of poliomyelitis. The explanation would be that the segment of the cord that has to do with the knee-reflexes is unaffected. He thought from his examination of the child that the quadriceps muscles were not in the same atrophied condition as those below the knee. He had seen a number of cases of poliomyelitis with retained knee-jerk.

Dr. A. A. Eshner stated that this patient had been at the Orthopedic Hospital a number of times, and it was recorded that the knee-jerks were "active." The records of this institution show that in many cases of poliomyelitis the knee-jerks are preserved, and in a few they are noted as pronounced. While exaggeration of the knee-jerk in,cases of poliomyelitis is uncommon, its preservation is by no means rare.

Carcinoma of the Spine with Autopsy.—This paper was read by Dr. A. Buckley.

Dr. William G. Spiller said he had recently had a similar case with parapelgia, considerable pain and loss of the reflexes. Carcinoma of the vertebræ secondary to carcinoma of the breast was found. Within a few weeks he had seen another similar case of tumor of the breast with symptoms of compression of the cord, due to secondary growth in the vertebræ.

Dr. Wharton Sinkler said he had seen a case of carcinoma of the cord following carcinoma of the breast. The cord symptoms appeared about three months after excision of the breast. The lesion was evidently high in the cord as paralysis of the arms as well as of the legs was an early symptom. The patient lived three months after the development of the cord symptoms. No autopsy was obtained.

Dr. A. C. Buckley said that throughout the lumbar cord there was a marked increase in the amount of neuroglia. The nerve roots did not stain as other nerve roots. There was round-cell infiltration among the nerve fibers. The ganglion cells of the gray matter of the lumbar cord were very much affected.